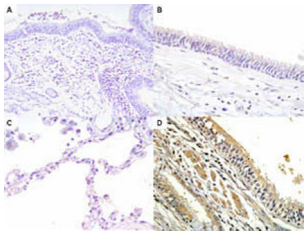
Methods: We assessed the expression patterns of CRBP-1 in 84 stage I non-small cell lung cancer (NSCLC), with the aim of characterizing their interrelationship via immunohistochemical staining (IHC) and survival.



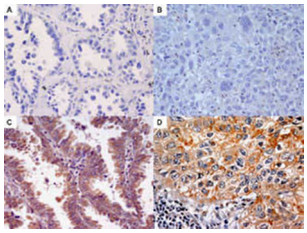


Figure 1. Expression of CRBP-1 in normal epithelial and alveolar cells

A, B, C- negative, D.focal positive.

Figure 2. Expression of CRBP-1 in stage I non-small cell lung cancer cells, A and B- negative in adenoca and squamous cell ca, C and D- positive in adenoca and squamous cell ca.

Results: The patients consisted of 65 men and 19 women. The mean age of these patients was 62 ± 10 years and 60 (71%) were smokers. Histologic subtypes included 40 cases of adenocarcinoma, 40 of squamous cell carcinoma, and 4 of large cell carcinoma. Fourteen out of 84 (16.7%) cases exhibited positive expression of CRBP-1 in more than 25% of tumor cells. The sex, smoking status, tumor size, histologic types, and differentiation were not statistically different in the presence of CRBP-1 expression. In Kaplan-Meier survival analysis, expression of CRBP-1 proved to be a statistically significant poor prognosis factor in overall survival (p=0.026). Cox regression analysis showed that the expression of CRBP-1 seemed to be associated with poor prognosis factor in stage I NSCLC (p=0.006).

Conclusions: CRBP-1 expression may play a partly role in carcinogenesis and prognosis of early stage lung cancer. Further prospective studies are warranted to determine the molecular mechanism in regard to the role of CRBP-1 in lung carcinogenesis.

P2-091 **BSTB: Prognostic Factors Posters, Tue, Sept 4**

Can protein expression, polymorphism and mutation of VGEF gene predict the response of targeted therapy in NSCLC lung cancers?

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Background: Vascular Endothelial Growth Factor (VEGF) is known to play an important role in angiogenic process of lung cancers. Whether the level of VEGF expression in cancers will reflect the response of Anti-VEGF treatment, no one know. We seek to answer two questions:

1) Can VEGF protein expression predict the lung cancer response from Anti-VEGF treatment?
2) Can Sequence Variation and Mutations of VEGF gene predict the tumor response In NSCLC lung cancer treatment?

Methods: Included in the study were 28 cases of NSCLC Lung cancers. We use Immunohistochemical (IHC) for VEGF protein expression. Their Tumor DNA were extracted and studied for polymorphism and mutations of VEGF gene, using DNA sequencing technique.

Results:

1) Lung NSCLC showed 70% (20/28) VEGF protein expression. With 8 cases of strongly expressed and 12 cases of low expressed VEGF protein. DNA sequence of VEGF gene showed no mutations in 8 cases of strongly expressed VEGF and 11 cases of low expressed VEGF protein. VEGF gene mutations occurred in VEGF negative at the rate of 3/8 and 1/12 in low expressed VEGF protein. The mutation of VEGF gene in NSCLC lung cancer occurred at the rate of 4/28 = 14 %
2) Polymorphism study in all positive VEGF appeared to suggest that weakly expressed protein had genotype codon 108 AA with less tumor aggressiveness. And genotype codon 108G/G and/or codon 108 G/A appeared to be associated with more aggressiveness.
3) All the NSCLC responders showed low expressed VEGF protein or VEGF negative protein

Conclusion:

1) Tumors with VEGF protein positive did not have any VEGF gene mutations.
2) It appeared that only VEGF protein negative tumors showed mutations in their VEGF gene.
3) Low VEGF protein expression and No VEGF protein expression appeared to do well and survived longer after treatment. The level of VEGF protein expression and their relationship with EGFR and KRAS mutations in our NSCLC lung cancers will be included in our presentation.

P2-092 **BSTB: Prognostic Factors Posters, Tue, Sept 4**

14-3-3 sigma and checkpoint with forkhead and ring finger (CHFR) methylation in serum in erlotinib-treated non-small-cell lung cancer (NSCLC) patients (p) with EGFR mutations

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Background: 14-3-3 proteins have 130 potential binding partners, including Cbl. 14-3-3 expression can prevent mutant EGFR binding to Cbl, impairing ubiquitination and endocytosis. 14-3-3 σ is frequently methylated in NSCLC; we hypothesized that in the presence of EGFR mutations, methylated 14-3-3 σ could permit the formation of the EGFR-Cbl complex. CHFR is a checkpoint that delays entry into metaphase in response to mitotic stress.

Methods: 73 stage IV NSCLC p with EGFR exon 19 deletion or exon 21 L858R mutation received first- or second-line erlotinib single therapy. 14-3-3σ and CHFR methylation was examined in the baseline serum of these p.

Results: Median age, 63 (range, 26-83); females, 48 p (65.8%); Caucasian, 72 p, Asian, 1 p; never-smokers, 45 p, ex-smokers, 21 p, smokers, 7 p; adenocarcinoma, 64 p, large cell carcinoma, 9. PS: 0, 19 p, 1, 42 p, 2-3, 12 p. 14-3-3σ was methylated in 39.7% and CHFR in 42.5% of p. No differences in p characteristics were observed according to methylation status. Complete response was observed in 11.1% of p, and partial response in 75.4%. Overall response was 86.5%. There was a trend toward a higher response rate in p with unmethylated CHFR (94.4% vs 76.6%; P=ns). Overall median time to progression (TTP) and survival